SPOTLIGHT

Articles of Significant Interest Selected from This Issue by the Editors

LipL32 Is an Extracellular Matrix-Interacting Protein in Diverse Bacteria

LipL32 is the major surface protein of pathogenic *Leptospira* spp., and its function has eluded researchers for almost a decade. Hoke et al. (p. 2063–2069) now show this protein to interact specifically with the extracellular matrix. Although unique among spirochetes, Lip32 has a homolog in the taxonomically distinct marine bacterium *Pseudoalteromonas tunicata*. Surprisingly, the homolog was also found to bind mammalian extracellular matrix. Thus, these distinctly different bacteria appear to interact with vastly different hosts through similar mechanisms.

Addressing Regulatory T Cells: Beneficial for Intestinal Nematodes?

Regulatory T cells (Tregs) have recently evoked much interest in the context of chronic parasite infections. Investigating the role of Tregs and effector T cells during infection with the roundworm *Heligmosomoides polygyrus*, Rausch et al. (p. 1908–1919) show distinct changes in the phenotype and distribution of Tregs and endorse the view of CD4⁺ T cells as being essential for protection. Adoptive transfers of specific T effector cells mediated protection, while transfer of Tregs did not influence worm burden. Furthermore, cotransfer of both subsets revealed that Tregs failed to control the antiparasitic virtue of effectors. Thus, the role of Tregs as beneficial to parasites is brought into question.

Leishmania Amastigote Surface Glyconjugate That Is Recognized by Toll-Like Receptor 4

Toll-like receptors (TLRs) are important in pathogen recognition and subsequent induction of inflammatory responses. While several studies suggest a role for TLRs in the recognition of the *Leishmania* promastigote stage, the importance of TLRs to the amastigote stage found in the mammalian host is not clear. Work by Whitaker et al. (p. 2149–2156) shows that *Leishmania pifanoi* amastigotes and an amastigote-derived glyconjugate (the P8 proteoglycolipid complex [P8 PGLC]) can induce inflammatory immune responses through Toll-like receptor 4 (TLR4). Furthermore, lack of TLR4 led to exacerbated infection in *L. pifanoi* amastigote-infected mice. These findings establish an important role for TLR4 recognition of *L. pifanoi* amastigotes in part through the P8 PGLC.

Aspergillus fumigatus DNA Stimulates Toll-Like Receptor 9-Dependent Responses

Wild-type and Toll-like receptor 9 (TLR9) knockout mice differ in their susceptibilities to aspergillosis. As TLR9 is an endosomal DNA sensor, Ramirez-Ortiz et al. (p. 2123–2129) looked at the capacity of human and murine dendritic cells to respond to *Aspergillus fumigatus* DNA. The cells secreted proinflammatory cytokines when stimulated by fungal DNA and by synthetic oligodeoxynucleotides containing sequences in the *A. fumigatus* genome. Optimal responses required TLR9 on host cells and unmethylated cytosine guanine dinucleotides in the fungal DNA. This study demonstrates a novel way by which the innate immune system may sense *A. fumigatus*.